EXHIBIT 1002
Superficial temporal artery to middle cerebral artery anastomosis

Intraoperative evaluation by fluorescein angiography and xenon-133 clearance

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Fluorescein angiography and xenon-133 (133Xe) clearance studies were performed during surgery on 15 patients who were undergoing superficial temporal artery (STA) to middle cerebral artery (MCA) anastomosis. Fourteen patients had occlusive disease of the internal carotid artery (ICA), and one patient had severe stenosis of the MCA. Before anastomosis, fluorescein angiography showed slow filling of the MCA branches through collateral channels. Focal areas of impaired microcirculatory filling and washout were seen in the territory of severely sclerotic cortical arteries. The findings of preanastomotic 133Xe clearance studies were variable and a uniform pattern of regional cerebral blood flow (rCBF) changes was not defined. In 55% of the patients, rCBF was reduced to 25 ml/100 gm/min or less at one or more detector sites. Fluorescein angiography provided an immediate assessment of anastomotic patency and clearly displayed the distribution of blood entering the epicerebral circulation through the STA. In 67% of patients, multiple MCA cortical branches filled with fluorescein, whereas in 33% filling was restricted to the receptor artery territory. An immediate, substantial (> 15 ml/100 gm/min) increase in rCBF was demonstrated in 73% of patients after anastomosis. The rCBF changes were consistently better in patients with donor and receptor arteries greater than 1 mm in diameter. Redistribution of collateral input acted to increase rCBF in areas distant from the anastomotic site. Some improvement in fluorescein circulation and rCBF also was seen in cortex supplied by sclerotic MCA branches.

KEY WORDS • cerebral ischemia • cerebral revascularization • cerebral blood flow • fluorescein angiography

Cerebral blood flow (CBF) in patients undergoing superficial temporal artery (STA) to middle cerebral artery (MCA) anastomosis has been studied before and after surgery by Schmiedek, et al.,18 Yamamoto, et al.,12,23,24 and others.1,14 These investigations demonstrated reduced regional cerebral blood flow (rCBF), usually multifocal, in the cerebral hemisphere ipsilateral to the occluded internal carotid artery (ICA) or MCA. The revascularization procedure consistently improved rCBF, and symptoms of cerebral ischemia invariably did not recur.

Information about the changes in the epicerebral circulation and rCBF during surgery is limited. Experimental studies have been performed on dogs,16,16 but the results, although interesting, are difficult to relate to the human situation. The object of this investigation was to study the epicerebral circulation and rCBF during surgery in patients undergoing STA-MCA anastomosis for occlusive disease of the ICA or MCA. This was accomplished using fluorescein angiography and the xenon-133 (133Xe) clearance technique.
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### TABLE 1
Clinical presentation and diagnostic findings in 15 patients undergoing STA-MCA anastomosis*

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Sex, Age (yrs)</th>
<th>Clinical Diagnosis</th>
<th>Clinical Localization of Cerebral Ischemia</th>
<th>Angiographic Findings</th>
<th>CT Scan Findings</th>
</tr>
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<tbody>
<tr>
<td>1</td>
<td>M, 44</td>
<td>recent small stroke, TIA'S</td>
<td>rt frontal, parietal</td>
<td>rt ICA stenosis</td>
<td>no abnormality</td>
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<td>2</td>
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<td>recent small stroke, TIA'S</td>
<td>lt frontal, lt ICA occlusion, lt ECA stenosis (95%), rt CCA occlusion</td>
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</tr>
<tr>
<td>3</td>
<td>M, 68</td>
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<td>lt parietal, lt ECA stenosis (95%)</td>
<td>small lt cerebral infarction</td>
<td></td>
</tr>
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<td>4</td>
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<td>recent small stroke, amaurosis fugax</td>
<td>lt parietal, rt &amp; lt ECA occlusion, lt ICA stenosis (50%)</td>
<td>small lt cerebral infarction</td>
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<td>rt frontal, rt ICA occlusion</td>
<td>small rt cerebral infarction</td>
<td></td>
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<tr>
<td>6</td>
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<td>recent small stroke</td>
<td>lt parietal, lt ECA stenosis, rt frontal</td>
<td>bilateral cerebral infarction</td>
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<tr>
<td>7</td>
<td>F, 60</td>
<td>recent small stroke, TIA'S</td>
<td>lt frontal, lt ICA stenosis (suprachinoid)</td>
<td>no abnormality</td>
<td></td>
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<tr>
<td>8</td>
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<td>old lt cerebral stroke, TIA'S</td>
<td>rt frontal, rt ECA stenosis (70%)</td>
<td>large lt cerebral infarction (old)</td>
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<tr>
<td>9</td>
<td>F, 61</td>
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<td>rt frontal, rt ECA occlusion, rt frontal</td>
<td>small rt cerebral infarction</td>
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<tr>
<td>10</td>
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<td>recent small stroke, TIA'S</td>
<td>rt frontal, rt ICA stenosis (50%)</td>
<td>small rt cerebral infarction</td>
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<td>11</td>
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<td>rt frontal, rt ICA stenosis (suprachinoid)</td>
<td>no abnormality</td>
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</tr>
<tr>
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<td>F, 47</td>
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<td>lt frontal, lt ICA stenosis</td>
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<td>13</td>
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<td>TIA'S</td>
<td>lt frontal, lt ICA stenosis</td>
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<td>lt frontal, parietal</td>
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<td>recent small stroke, TIA'S</td>
<td>lt frontal, lt MCA stenosis</td>
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</table>

*STA = superficial temporal artery; MCA = middle cerebral artery; TIA = transient ischemic attacks; ICA = internal carotid artery; ECA = external carotid artery; CCA = common carotid artery; CT = computerized tomography.

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**Clinical Material and Methods**

**Patient Population**

Fluorescein angiography and 133Xe clearance studies were performed during surgery on 15 patients (Table 1) undergoing STA to MCA anastomosis. The 11 men and 4 women ranged in age from 16 to 68 years (mean: 51 years). Atherosclerosis was the primary pathological process in the 14 adults. The arteriopathy in the 16-year-old male patient (Case 10) was thought to be the result of homocystinuria.

**Clinical Presentation**

Eleven patients presented with cerebral transient ischemic attacks (TIA's) in the MCA distribution. Nine of them had also suffered a recent (< 3 months ago) small cerebral infarct. One patient (Case 4) had recurrent amaurosis fugax and had suffered a recent small, ipsilateral infarct. The three remaining patients had experienced recent cerebral infarction only.

**Cerebral Angiography**

Preoperative angiography with visualization of the aortic arch, vertebral arteries, and carotid arteries (both intracranially and extracranially) was carried out in each case. Ipsilateral occlusion of the ICA was demonstrated in 11 patients. Inaccessible ICA stenosis was seen in three patients, including the 16-year-old boy (Case 10) who was shown to have progressive, severe stenosis of the supraclinoid segment. Four patients with ICA occlusive disease also had focal, high-grade (> 90%) stenosis at the origin of the ipsilateral external carotid artery (ECA). They underwent external carotid endarterectomy with insertion of a vein-patch graft 2 weeks before craniotomy. Another patient (Case 8) with a 70% focal stenosis of the proximal ECA had an external carotid endarterec-
tomy performed 2 weeks after craniotomy. Severe, focal stenosis of the MCA trunk was demonstrated in Case 15.

Operative Procedure

A No. 18 polyethylene catheter was inserted percutaneously into the ipsilateral common carotid artery (CCA) immediately before surgery. The catheter was intermittently irrigated with small amounts of heparinized saline (1000 units/500 ml isotonic saline) throughout the operation to prevent thrombus formation and to maintain catheter patency.

An inverted U-shaped scalp flap was turned. The larger STA branch, together with a generous cuff of connective tissue, was mobilized carefully. A relatively large temporoparietal craniotomy was performed. The largest exposed cortical artery, usually the angular branch of the MCA, was selected as the receptor vessel. Mean STA diameter was 1.3 mm (range: 0.9 to 1.6 mm) and mean receptor artery diameter was 1.2 mm (range: 0.9 to 1.6 mm). Continuous suturing was performed for the anastomosis in all but the initial three patients, in whom the standard interrupted suture technique was used. The operations were carried out by one surgeon (J.R.L.).

The arterial blood pressure was maintained at preoperative levels during the intraoperative studies. Arterial pCO2 was kept in the 40 ± 3 torr range.

Intraoperative Studies

Fluorescein Angiography. The technique of fluorescein angiography has been described in detail elsewhere. Studies were performed before and after anastomosis. Sodium fluorescein (5 ml of a 1% solution) was injected rapidly into the ipsilateral CCA through the indwelling catheter. Illumination for photography was provided by a strobe light equipped with a Kodak-Wratten 47A filter. Barrier filters (Kodak-Wratten 2B and 21) were used to keep unwanted exciting radiation from reaching the film. Rapid, serial photographs of the cortex were taken with a motorized Nikon camera fitted with a 200-mm Medical Nikkor lens. A data-back digital timer automatically printed the time in one-hundredths of a second in the corner of each frame. The surgeon viewed the operative field through a presterilized Kodak-Wratten 21 filter.

Xenon-133 Clearance Studies. Clearance studies were performed immediately following fluorescein angiography, that is, before and after anastomosis. The cortex was covered with a thin plastic film. Four small, lithium-drifted semiconductor detectors were placed gently on the plastic film overlying the inferior frontal, supramarginal, angular, and superior temporal gyri. Xenon-133 (10 to 12 mCi) dissolved in 3 ml of isotonic saline was rapidly injected into the ipsilateral CCA through the indwelling catheter. The detecting system was remotely connected through a scanner interface to a PDP-12 computer. The mean rCBF from each cortical area was calculated by a modification of the stochastic analysis.

Results

Intraoperative Findings

Gross Observations. Evidence of previous infarction, consisting of gyral atrophy and pallor, was seen in 10 patients. It involved a single gyrus in two patients, two or three gyri in six patients, and more than three gyri in two patients. In eight of these 10 patients, cortical infarction was found to lie in the territory of a severely sclerotic cortical artery. Neovascularization, consisting of numerous irregular, thin-walled vessels, was seen adjacent to the infarcted areas in 5 patients.

Fluorescein Angiography. Studies performed before anastomosis showed delayed filling of the cortical branches of the MCA. The mean duration between injection of fluorescein into the ipsilateral CCA and its initial appearance in the epicerebral circulation was 2.4 ± 0.4 seconds, compared with 0.7 ± 0.3 seconds following anastomosis. In patients with occlusive disease of the ICA, filling of the MCA branches occurred in an anterograde direction. In the patient with high-grade stenosis of the MCA trunk (Case 15), filling of the MCA branches occurred in a retrograde direction through epicerebral collateral channels from the anterior and posterior cerebral arteries.

Patency of the anastomosis was demonstrated in 13 patients. In one of these patients (Case 13), partial obstruction of the STA was seen at the site where the temporary occluding clip had been applied. This was corrected by gentle manipulation and the application of a small amount (< 2 ml) of 1% Xylocaine (lidocaine). The anastomosis was found to be occluded in two patients. A thrombus was successfully removed and patency restored in one of these patients (Case 6). In the other (Case 3), patency was re-established in the proximal segment only of the receptor artery. The cortical receptor artery in both patients was narrow (< 1 mm) and diffusely atherosclerotic.

Fluorescein transit time through the STA was slow (> 1.5 seconds) in two patients. One (Case 8) had a 70% stenosis at the origin of the ECA. The other patient (Case 9) had a diffusely narrow (< 1 mm) atherosclerotic STA.

Circulation time, that is, time between maximum arterial and venous filling, was improved in all
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Fig. 1. Fluorescein angiography after anastomosis in Case 4. Upper Left: Left temporoparietal craniotomy before fluorescein angiography. The superficial temporal artery was covered by a generous cuff of connective tissue and fat (X). Upper Right: At 01:13 seconds following injection of fluorescein, filling of the cortical receptor artery was observed. Center Left: The cortical branches of the middle cerebral artery filled in an anterograde direction. The microcirculation supplied by the receptor artery also has filled. Center Right: Microcirculatory filling was widespread. Lower Left: Filling of cortical veins has begun. Lower Right: At 11:14 seconds, the veins were well filled and microcirculatory washout was essentially complete. Leakage of fluorescein into the extravascular compartment was seen adjacent to the cortical receptor artery.
Fig. 2. Fluorescein angiography after anastomosis in Case 5. Upper Left: Right temporoparietal craniotomy before fluorescein angiography. The letters indicate the sites of the $^{133}$Xe detectors. Upper Right: At 00:86 seconds following injection of fluorescein, filling of the cortical receptor artery was observed. Lower Left: Fluorescein filling was limited predominantly to the receptor artery territory. Lower Right: At 04:74 seconds, the veins draining this area filled with fluorescein. Washout in the lower gyri was more rapid. Preanastomotic regional blood flow (rCBF), in ml/100 gm/min, at the four detector sites was: A, 15; B, 15; C, 17; and D, 20. Postanastomotic rCBF was: A, 56; B, 33; C, 60; and D, 40.

patients following anastomosis. Comparison of the preanastomotic and postanastomotic circulation times revealed a mean improvement of 44% (range: 10% to 80%).

Fluorescein angiography showed the distribution of blood supplied by the STA through the anastomosis. Of the 14 patients who underwent surgery for occlusive disease of the ICA, nine had filling of multiple MCA cortical branches (Fig. 1) and five had filling predominantly in the receptor artery territory (Fig. 2). Essentially all of the MCA cortical branches filled in an anterograde direction in the patient (Case 15) with MCA occlusive disease (Fig. 3).

Many of the severely sclerotic cortical arteries were occluded and consequently did not fill with fluorescein. The cortical microcirculation in their territory occasionally filled slowly through multiple irregular, thin-walled channels (that is, by neovascularity) coming from adjacent cortical arteries. Impaired microcirculatory filling and washout were often seen in areas supplied by patent, sclerotic MCA branches (Fig. 4). Although these regional flow abnormalities persisted after anastomosis, some improvement was noted.

Extravasation of fluorescein out of the microvasculature was occasionally observed adjacent to the
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Fig. 3. Fluorescein angiography after anastomosis in a patient (Case 15) with severe stenosis of the left middle cerebral artery (MCA). Upper Left: Left temporoparietal craniotomy prior to fluorescein angiography. Upper Right: At 01:37 seconds following injection of fluorescein, filling of the cortical receptor artery was observed. Center Left: The MCA cortical branches filled in an anterograde direction. Center Right: Microcirculatory filling was widespread. Lower Left: Microcirculatory washout and early venous filling were seen at 07:72 seconds. Lower Right: The cortical veins were filled with fluorescein at 12:69 seconds.

anastomotic site. Similar leakage was not seen at other locations.

Xenon-133 Clearance Studies. The results of the preanastomotic and postanastomotic studies are listed in Table 2. The preanastomotic rCBF values were similar (difference ≤ 10 ml/100 gm/min) at the four recording sites in nine patients. In the others, considerable variation was seen from one recording site to the next with areas of very low flow (≤ 25 ml/100 gm/min) interspersed with areas of more normal flow (≥ 40 ml/100 gm/min). Severe reduction of rCBF (≤ 25 ml/100 gm/min) at one or more recording sites was observed in eight patients.

Immediate, substantial (≥ 15 ml/100 gm/min) increases in rCBF were demonstrated following anastomosis in 11 patients. In four of them, the im-
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Fig. 4. Right temporoparietal craniotomy after anastomosis in Case 9. Left: Severe sclerosis of cortical arteries (arrows) was observed. Neovascular channels have developed in the vicinity of these diseased vessels. The STA was diffusely atherosclerotic and narrow (< 1 mm). It was obscured by a cuff of connective tissue and fat (X). Right: Fluorescein angiogram at 08:29 seconds showed slow arterial filling restricted to the receptor artery territory. The sclerotic cortical artery (arrow) filled poorly, and there was delayed microcirculatory filling of the cortex it supplied. Leakage of fluorescein into the wall of the STA and surrounding connective tissue was noted.

Improvement was predominantly in the receptor artery territory, whereas in the other seven patients, the improvement was generalized. The areas with increased rCBF did not always correlate with the distribution of fluorescein entering the epicerebral circulation through the STA. Mean improvement in rCBF was 12 ± 5 ml/100 gm/min in the patients with an STA and cortical receptor artery greater than 1 mm in diameter. The rCBF increase averaged 7 ± 4 ml/100 gm/min in those patients with an STA and/or cortical receptor artery diameter of 1 mm or less.

One patient (Case 10), who showed no increase in rCBF, had normal preanastomotic values (52 ± 1 ml/100 gm/min). Another patient (Case 3), with postanastomotic occlusion of the distal limb of the receptor artery, was found to have further reduction in rCBF in the area it supplied. The rCBF was not improved in the two patients (Cases 8 and 9) with slow STA fluorescein transit.

Clinical Course

The neurological status in the patient (Case 3) with the partially occluded anastomosis was slightly worse postoperatively. Another patient (Case 7) experienced a single TIA consisting of right upper extremity weakness and expressive dysphasia during an episode of orthostatic hypotension (systolic blood pressure < 100 mm Hg). The postoperative angiogram in this patient showed an occluded left ICA instead of a severe supraclinoid stenosis as demonstrated preoperatively. The 13 other patients were neurologically unchanged. There were no non-neurological complications.

Patency of the anastomosis was demonstrated on the 12 postoperative angiograms performed 10 to 14 days following surgery. An angiogram was not done on the patient (Case 3) with the partially occluded anastomosis shown by fluorescein angiography. Increase in the luminal diameter of the STA was observed in 11 patients (mean increase: 1.5 ± 0.5 mm). Filling of multiple MCA branches was seen in nine patients. One patient (Case 9) was found to have slow filling of the STA and cortical receptor artery. The luminal diameter of the diffusely narrow (< 1 mm), atherosclerotic STA in this case was unchanged from preoperatively.

All patients are alive 4 to 21 months (mean: 16 months) following surgery. Symptoms of cerebral ischemia have not recurred.

Discussion

Intraoperative fluorescein angiography and $^{133}$Xe clearance studies have been used extensively by us in...
the surgical treatment of cerebral arteriovenous malformations, and more recently in STA-MCA anastomosis. Information from the studies performed during the revascularization procedure has provided valuable insight into anastomotic patency, distribution of blood supplied by the STA, changes in epicerebral circulatory patterns, and rCBF. Permanent morbidity related to the use of these techniques was not encountered.

**Fluorescein Angiography**

Complete evaluation of collateral flow before anastomosis would have required catheterization of both CCA's and a vertebral artery. The added risk of such an investigation and the associated increase in surgical and anesthetic time were considered unacceptable.

Patients with ICA occlusive disease were found to have slow epicerebral filling and washout. Fluorescein injected into the ipsilateral CCA had to pass through collateral channels between the ECA and intracranial ICA before reaching the MCA branches. Initial filling of the cortical arteries consequently was delayed, and the concentration of fluorescein diluted. These findings suggested that the collateral supply from this source alone was inadequate.

Filling of the MCA branches before anastomosis occurred in an anterograde direction in patients with ICA occlusive disease. In the patient with MCA stenosis, filling of these branches was in a retrograde direction from the anterior and posterior cerebral arteries. A similar pattern of retrograde flow has been demonstrated in acute embolic MCA occlusion. Such a flow pattern is expected when the obstructed artery is distal to the circle of Willis, and underscores the collateral capacity of the epicerebral connections between the three major cerebral arteries.

Some variation of microcirculatory filling was observed. Focal impairment of fluorescein flow usually occurred in the territory of a severely sclerotic cortical artery. Adjacent cortex often appeared infarcted. Filling of the microcirculation in these areas occasionally took place through irregular, thin-walled neovascular channels arising from less-diseased cortical arteries. Filling and washout of fluorescein was slow but some improvement was observed following anastomosis. These findings indicated the importance of occlusive disease in the epicerebral arteries in patients with ICA or MCA occlusion.

Using the technique of fluorescein angiography described by Feindel, et al., and Meré,, performed studies on patients undergoing surgery for cerebral ischemia caused by thrombosis or stenosis of either the ICA or MCA. He described the frequent extravasation of fluorescein from veins in ischemic foci. Similar leakage of fluorescein into the extravascular compartment was not observed in our study. Some extravasation of fluorescein, however, was seen occasionally in the vicinity of the anastomotic site. This was thought to be the result of slight surgical trauma and temporary occlusion of the cortical receptor artery.

Verification of anastomotic patency during surgery is of utmost importance. Recent experimental studies by Rosenbaum and Sundt suggest that anastomoses which are patent at 30 minutes remain patent thereafter. Our findings substantiated this conclusion. It is unlikely that patency of the anastomosis can be re-established when the STA fails to fill on the post-operative angiogram unless it is performed shortly after surgery. Direct observation of the anastomosis alone can be misleading, as shown in three of our patients. All three anastomoses appeared to be functioning well when examined with the operating microscope; however, fluorescein angiography revealed thrombosis at the anastomotic site in two and obstruction of the STA at the site of temporary clip application in one. Satisfactory flow through the anastomosis subsequently was achieved in two of them.

Fluorescein angiography clearly displayed the distribution of blood entering the epicerebral circulation through the STA. Filling and washout of the microcirculation was generally improved. In 67% of patients, multiple cortical branches of the MCA filled with fluorescein, whereas in 33% only the receptor artery and its branches filled. Direction of flow in the cortical arteries of the patient with MCA stenosis had converted from retrograde to anterograde. The flow patterns observed in the 15 patients were thought to reflect the equilibrium established between the pre-existing collateral input and the newly created arterial source.

**Xenon-133 Clearance Studies**

The findings before anastomosis were somewhat variable, and a uniform pattern of rCBF changes was not defined. Multifocal reduction of rCBF, similar to that described by Schmiedek, et al., was not demonstrated consistently but foci of reduced rCBF could have been missed as only four detectors were used. Multifocal reduction of rCBF was demonstrated in 13 of our patients studied preoperatively with krypton-77 positron emission tomography. The results of these studies are reported separately.

Electroencephalographic abnormalities were observed by Sundt, et al., when rCBF was acutely reduced below 17 to 18 ml/100 gm/min in patients undergoing carotid endarterectomy. Other investigators have shown experimentally that infarction occurs if these low rCBF levels persist. In 55% of our patients, rCBF was reduced to 25 ml/100 gm/min or less at one or more detector sites.

The rCBF values invariably were below 25 ml/100 gm/min in areas supplied by severely sclerotic cortical arteries. It is not surprising that the adjacent cortex frequently had changes indicative of previous infarction. The presence of occlusive disease of the cortical arteries could be an important factor in producing the
A multicentric pattern of ischemia described in other reports. Improvement in rCBF was demonstrated in 73% of patients following anastomosis. Increased rCBF was not confined to the territory of the receptor artery in most patients. Mean rCBF increases were comparable at the four detector sites. Improvement in rCBF was frequently seen in areas supplied by sclerotic cortical arteries, although it tended to be less pronounced than in areas supplied by normal-appearing arteries. Failure to improve rCBF resulted from thrombosis at the anastomotic site, diffuse atherosclerotic narrowing of the STA or cortical receptor artery, or focal stenosis of the ECA. In one patient, normal rCBF values before anastomosis were unchanged postoperatively.

The rCBF was consistently better in patients with donor and receptor arteries greater than 1 mm in diameter. Chater4 has reported similar findings. Using a flowmeter attached to the STA during surgery, he showed a mean flow of 37 ± 15 ml/min in patients with arteries greater than 1 mm in diameter. Those patients with one or both arteries measuring 1 mm or less in diameter had a mean flow of 18 ± 8 ml/min. These findings stress the importance of utilizing the largest arteries available when performing the anastomosis.

Some incongruities between the findings of post-anastomotic fluorescein angiography and 133Xe clearance studies were seen. Substantial increases in rCBF were occasionally demonstrated in areas with little or no fluorescein filling. Arterial blood supplying these areas likely was derived from epicerebral collaterals from the anterior and posterior cerebral arteries as well as deeper cortical layers and subcortical white matter not visualized by fluorescein angiography. Collateral flow in these areas could be identified, however, by determining the clearance of the gamma-emitting 133Xe. The 133Xe clearance curves indicated a redistribution of blood flowing into the epicerebral circulation, resulting in improvement in rCBF that was more widespread than was evident with fluorescein angiography alone.

Clinical Considerations

Our clinical results substantiate previously reported studies indicating that STA-MCA anastomosis improves the natural history of selected patients suffering from cerebrovascular occlusive disease.1, 8, 20, 28 The findings of this study suggest that improved cerebral perfusion is an important factor in the prevention of cerebral infarction in these patients.

Acknowledgments

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References


7. Garretson H, Feindel W: Personal observation


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